who still feel this way are greatly overbalanced by the majority of birth parents eager to find these children.[4] In fact, many adoptees do not even seek out these parents, but are satisfied with information about their heritage. Persons working in the adoptive field use the term birth parents to distinguish this person (or persons) from the adoptive parents who are the true psychological and meaningful parents of the adoptee. Much of this was eloquently expressed in the reply by C. Mark Hynum in the February 1982 issue.[1] As an adoptee, he understands many of the problems of which much of the medical profession is unaware. Only recently has adoption come out of the closet and people in the adoption community are becoming able to express their feelings and their problems.

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Western Student Medical Research Forum

TO THE EDITOR: There is concern among medical educators that diminishing student interest in academia as a career threatens the quality of future medical education and the vitality of American clinical research.

It is said that among previous generations of medical students, about half expressed interest in academic careers, while today only a few are so inclined. The reasons for this are complex and include economic as well as other factors.

Measures currently being taken to counter this trend include increases in clinical investigator traineeship positions and support for medical student research.

One of the most vigorous and, I believe, successful efforts to encourage student research is being coordinated by a lay person. The Western Student Medical Research Forum (WSMRF) provides an opportunity for students to present their work in a scientific session held conjointly with the Annual Meeting of the Western Section of the American Federation for Clinical Research (AFCR). Representing the 16 western medical schools, the WSMRF has earned a remarkable reputation for quality and consistency among student research forums.

Since 1974 the WSMRF has been coordinated by Mrs. D. P. Bertakis of Sacramento, California. She became involved when her daughter, then a student, became chairman of the Forum. Mrs. Bertakis realized the need for expert administrative direction if this student-initiated effort was to thrive.

And thrive it did. The 1982 Forum saw more than 60 student papers presented, and had noted faculty representation throughout its two-day program.

Mrs. Bertakis has made a truly commendable contribution to medical education. Without remuneration, she annually spends considerable time organizing what may be the gem of medical student research forums. She has touched the lives and, I believe, influenced the career direction of medical students throughout the West. In addition, she may have shown us a particularly effective way to rekindle student interest in academia as a career.

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Heparin-Induced Thrombocytopenia

TO THE EDITOR: The article entitled "Risk of Complications During Intravenous Heparin Therapy"[1] by Nelson and co-workers in the March issue was well-written and informative. Unfortunately, the character of their series underestimates the incidence of heparin-induced thrombocytopenia (HIT) as a risk of heparin therapy, and their conclusions underemphasize its significance. Heparin-induced thrombocytopenia is a major complication of heparin therapy. In susceptible persons an immunologically mediated response causes increased platelet adhesiveness and aggregation.[2] Platelet-fibrin clot formation may follow, causing arterial or venous thromboembolism. Both large and small vessels may be occluded, including the abdominal aorta, sagittal sinus and coronary arteries. Clinical manifestations include limb ischemia, recurrent venous thrombosis, myocardial infarction and cerebrovascular accidents. When associated with arterial thromboembolism, the mortality of this complication may be as high as 60 percent.[3]

Heparin-induced thrombocytopenia is not limited to that population that Nelson and associates found to be most at risk for heparin complications

—that is, those above the age of 60 and those with concurrent disease. In 1977 we reported on three patients with heparin-induced thrombocytopenia.[4] None had concurrent disease, and their ages were 75, 49 and 31 years. The 31-year-old patient, receiving heparin therapy for a pulmonary embolism, had a thrombosis of the abdominal aorta with subsequent permanent paraparesis and ischemia of one leg requiring an above-the-knee amputation. The incidence of HIT reported in the literature varies widely. Although one prospective study showed an incidence of 31 percent,[5] most authors feel the true incidence is significantly lower than this figure. Nelson and coauthors admit that the incidence they report (1.5 percent) may be falsely low due to the lack of frequent platelet count determinations in their patients. However, in assessing the reported incidence of HIT, one must consider not only the frequency of platelet count determination but also the duration of heparin therapy. HIT occurs between 2 and 14 days after the onset of treatment.[5] Thus, only 107 of Nelson's patients may have been expected to be susceptible to this complication.

Although of low incidence, HIT may occur in any patient, including those of young age and without concurrent disease. Respecting the significant morbidity and mortality of this complication, we strongly recommend daily platelet count determination for all patients receiving heparin therapy.

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TO THE EDITOR: An important finding in the study by Nelson and co-workers[1] that deserves greater emphasis is that bleeding complications during heparin therapy do occur even when only modest doses of heparin are employed. It is sometimes argued that close monitoring or various in vitro coagulation tests will help prevent bleeding complications, presumably by avoiding excessive anticoagulation. What were the activated partial thromboplastin time (PTT) values in the patients who bled?

We reviewed the records of 121 patients with objectively documented deep vein thrombosis treated by continuous heparin infusion. Eleven patients (15 percent) had significant bleeding complications. The activated clotting time (ACT) was in the therapeutic range (150 to 190 seconds) in 9 of the 11, less than 150 seconds in 1 and greater than 190 seconds in the other. Of the 121 patients, 26 received "high dose" heparin therapy (150 units per kg of body weight and 30 to 40 units per kg of body weight per hour without regard to ACT levels) and the remainder, 95, received more conventional doses (50 units per kg bolus with subsequent infusion rates adjusted to achieve a therapeutic ACT). Significant bleeding occurred in 2 of 26 high-dose heparin cases and 9 of 95 conventional dose cases, an insignificant difference. The high-dose group had no recurrent thromboembolism whereas in the conventional-dose group there were eight documented cases of pulmonary embolism ($P=.047$), one of which was fatal.

Another point that deserves mention is the exercise of questionable judgment in giving heparin to patients with a high risk of intracranial bleeding. Few would argue against withholding heparin therapy in this group of patients. The judgment that must be made in each case is whether the risk of heparin therapy outweighs the risk of further thromboembolism. In most patients, as the authors have concluded, the risk of heparin therapy is minimal. In others, avoiding anticoagulation and employing vena cava interruption when indicated is the best course of action.

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Dr. Moser Replies

TO THE EDITOR: Dr. Baird's concerns about thrombocytopenia are certainly warranted. As we indicated, our investigation was not intended to define the incidence of thrombocytopenia or certain other events. Rather, we were interested in what was actually happening in a "slice of practice." One of the things that was *not* happening was daily platelet counts. Whether daily platelet